2. Acute effects of cigarette smoking in man detected by various techniques. Immediately after inhalation of cigarette smoke, there is an increase in airway resistance in normal subjects, both smokers and nonsmokers (Attinger et al., 1958; Sterling, 1967; Chiang and Wang, 1970; Reintjes et al., 1972; Da Silva and Hamosh, 1973). The bronchoconstriction was reduced by prior administration of atropine, indicating that the vagus nerve participates in the response (Sterling, 1967). Filtration of the smoke to remove either the particulate or the vapor phase had a similar effect in reducing the bronchoconstrictor response, showing that both phases contribute to this response (Clarke et al., 1970). Inhalation of isoproterenol prevented the response, which reflects its reversibility (Nadel and Comroe, 1961).

The acute bronchoconstriction has been reported following inhalation of cigarette smoke in patients with chronic lung disease: Simonsson (1962, 1965) reporting from Sweden; Cellereno and Billia (1962) from Italy; McDermott and Collins (1965) from Great Britain; Johannsen (1966) from Poland; James (1970) from Ohio; and Votchal et al. (1970) from the Soviet Union. These effects are transient in nature and do not necessarily contribute to the abnormal ventilatory function.

3. Acute and chronic effects of inhalation of cigarette smoke in animals. The author of this review and his collaborators reported the effects of inhalation of cigarette smoke in several animal species:

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VI-B. AIRWAY RESISTANCE

That cigarette smoking reduces forced expiratory volume in certain individuals described in the preceding section indicates a bronchospastic component. The other techniques used to investigate airway resistance are discussed in this section.

1. Body plethysmograph technique. The measurement of airway resistance or airway conductance by body plethysmograph technique is more sensitive than others applicable to man. All reports uniformly show an increase in resistance or a decrease in conductance in smokers as compared with nonsmokers: Pelzer and Thomson (1966) reporting from England on 82 subjects 17 to 82 years of age; Sevčik (1967) from Czechoslovakia on 150 men 18 to 68 years of age; Guyatt and Alpers (1968) and Guyatt et al. (1970) from England on 752 men 38 to 67 years old; Allen and Sabin (1971) from Texas on 32 males and females 16 to 82 years old; Diamond et al. (1971) from Kentucky on 30 subjects 19 to 44 years old; Huttemann et al. (1971, 1972) from Germany on 20 subjects 20 to 40 years old. The extent of increase in resistance varies with the age of the individual and the country of origin. The bronchoconstriction is readily reversed by abstinence from smoking for a few days (Cohen, 1966; Wilhelmsen, 1967; McFadden and Linden, 1972) or by the inhalation of a bronchodilator drug (Curtis et al., 1966; Crompton, 1968; Woolcock et al., 1969; Ruff et al., 1972).

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dog (Aviado and Palecek, 1967; Klide and Aviado, 1968; Cho et al., 1968; Carrillo and Aviado, 1970); cat (Palecek and Aviado, 1967); rabbit (Palecek et al., 1967); and rat (Aviado and Carrillo, 1969; Shore and Aviado, 1969; Aviado et al., 1970). The bronchoconstriction is brought about by the following mechanisms: (a) stimulation of cholinergic receptors in the airways; (b) stimulation of sensory receptors in the lungs, which triggers reflex spasm and (c) release of histamine, which produces bronchospasm (Aviado and Samanek, 1965; Samanek and Aviado, 1965; Samanek et al., 1965). Simultaneously, bronchodilatation is also produced by stimulation of adrenergic mechanisms arising from nicotine contained in the cigarette smoke and by reflex mechanism.

Following chronic exposure to cigarette smoke, there is an increase in airway resistance in rats (Ito and Aviado, 1968; Aviado, 1972) and mice (Aviado, 1972). This effect is reversed by inhalation of sympathomimetic drugs. The results in animals serve to explain the reversibility of bronchoconstriction as seen in man and described in the above paragraphs.

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A similar hypoxemia was observed to arise from smoking by patients with chronic lung disease in Ireland (Chapman, 1965) by patients with chronic bronchitis in England (Penman and Howard, 1966), and by patients with chronic bronchitis in Massachusetts (Strieder and Kazemi, 1967; Strieder et al., 1968). Hypoxemia is characteristic of patients in whom it does not necessarily relate to smoking but to pathological lesions in the lung (Pain et al., 1967; Weiss and Dulfano, 1968; Poppius, 1969).

Postoperative complications are reported to be higher among nonsmokers than smokers in Great Britain (Morton, 1944; Piper, 1958; Wightman, 1968). Smokers from Sweden do not show a higher incidence of pulmonary postoperative complications (Wiklander and Norlin, 1957). It has been suggested that postoperative hypoxemia is related to the appearance of pulmonary complications among smokers in Australia (Morton, 1969).

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15 to 18 years of age (Seely et al., 1971). However, the age and height of smokers and nonsmokers were not matched, so that it is not possible to specify the age when smoking influences ventilatory function.

The technique of pairing a smoker with a nonsmoker of the same age and sex has been used by two groups of investigators. Wilson et al. (1960) reported 14 pairs in Texas all over 40 years old and noted a decrease in timed expiratory volume, total lung capacity and maximal breathing capacity. Carey et al. (1967) reported 7 pairs in Ireland ranging from 19 to 60 years of age. Ventilatory capacity measured daily was less in smokers than in nonsmokers. The size of the group does not permit the conclusion as to the age when smoking starts to influence ventilatory capacity.

4. Abstinence from smoking. In some smokers with abnormal ventilatory function, cessation of smoking caused an improvement (Peters and Miller, 1960; Wynder et al., 1967; Peterson et al., 1968; Schuman, 1971). Administration of bronchodilator drugs has also reduced the airway obstruction in smokers (Petty et al., 1970). The abnormalities in ventilatory function associated with smoking are reversible.

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The functional activity of the ciliated epithelium can be influenced by oxygen tension (Dalhamn and Rosengren, 1968), particulate matter (Falke et al., 1959), and acetylcholine (Kordek et al., 1952). Cigarette smoke can exert its influence through any one or more of these mechanisms and this may account for the varied results reported in the rabbit. Kensler and Battista (1963), by using smoke filters, concluded that the ciliotoxic activity is in the gaseous phase. The following constituents of smoke depress ciliary activity in the rabbit: carboxylic acid and aldehydes (Wynder et al., 1965), phenol (Dalhamn, 1966) and acrolein (Scharbort, 1967).

3. Mucociliary transport in the cat. The observations in the cat have consistently shown a depression of bronchial clearance by exposure to cigarette smoke (Dalhamn, 1964, 1966; Carson et al., 1966; Kaminski et al., 1968). The tar content of the cigarette smoke influenced its ciliotoxicity (Nakashima, 1938; Dalhamn and Rylander, 1964, 1965, 1967, 1968). This effect was not simulated by nicotine but could be prevented by oxolamine citrate (Dalhamn, 1968, 1969). The role of the volatile constituents in cigarette smoke has been debated (Kensler and Battista, 1966; Dalhamn and Rylander, 1967, 1970; Kensler, 1967). Some chemical constituents of cigarette smoke have been shown to interact in their effect on mucociliary transport, which might explain the varied action reported for cigarette smoke (Weissbecker et al., 1971).

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VI-C. BRONCHIAL CLEARANCE

Cigarette smoking retards the clearance mechanism in certain experimental conditions. The effects of cigarette smoking on ciliary motility and mucus secretion are part of the defense mechanisms in the lung against inhalants in general and air pollutants in particular. (Gross, 1967; Gross and Hatch, 1963; Kilburn, 1967, 1968).

1. Mucociliary transport in man. The first observations relating to the effect of cigarette smoking on the respiratory epithelium were made in vitro (Rakieten et al., 1912). Strips of the nasal mucosa were examined under the microscope. The solution through which cigarette smoke was passed did not influence ciliary activity for a 5- to 10-min period. Longer exposure caused cessation of ciliary activity. These results were difficult to relate to the usual manner of smoking. Ballenger (1960) introduced another in vitro technique, using fresh mucosa scraped under general anesthesia from the trachea of children. The ciliotoxicity from the smoked solution is also difficult to relate to the usual form of smoking.

The introduction of radioactive particles for measurement of bronchial clearance has made it possible to investigate the influence of cigarette smoking in man. The results so far have not been consistent. Albert et al. (1969a) used monodisperse ferric oxide aerosols (0.8 to 7.9 μ): bronchial clearance was impaired in 8 out of 14 cigarette smokers. Pavia et al. (1970, 1971) and Thomson and Pavia (1973) used radioactive polystyrene particles 5 μ in diameter. There was no demonstrable difference in mucociliary transport between smokers

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and nonsmokers. Lourence et al. (1971) used particles 2 μ in size and noted that the clearance was delayed in smokers as compared with nonsmokers. Camner and Philipson (1971) and Camner et al. (1971, 1973) used 6 to 7 μ particles and observed a faster clearance immediately following smoking. However, habitual smokers showed an increase in clearance three weeks after abstaining. There appear to be two kinds of effects produced by cigarette smoking, an acute type which accelerates and a chronic type which decelerates clearance. Mucociliary transport in the nasal cavity is either retarded by smoking (Evert, 1965) or not influenced by it (Quinlan et al., 1969).

In 10 monozygotic pairs of twins that were discordant with regard to cigarette smoking, half of the smoking pairs had a markedly slower clearance than the nonsmokers, whereas the other half had a similar clearance (Camner and Philipson, 1972; Camner et al., 1972). It has not been possible to obtain a consistent effect from cigarette smoking even by comparing a smoker with a nonsmoker who is constitutionally similar.

2. Mucociliary transport in the rabbit. The results in animals are also variable. There was no effect from two hours' exposure to cigarette smoke on ciliary activity in vivo in the rabbit (Dalhamn, 1964) and on bronchial clearance of radioactive particles in the same species (LaBelle et al., 1966). A depression has been reported by others, using different techniques for measuring ciliary activity (Krueger and Smith, 1958) and clearance (Holma, 1969).

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4. Mucociliary transport in other animal species. The following animal species have been used in investigation of the influence of cigarette smoke: rat (Dalhamn and Rhodin, 1956; Dalhamn, 1959; Ferin et al., 1966; Guillerm et al., 1961); guinea pig (Hilding and Filipi, 1966); cow (Hill, 1928; Mendenhall and Shreeve, 1937; Hilding, 1956 a, b); monkey (Kensler and Battista, 1966); chicken (Battista and Kensler, 1970 a, b); donkey (Albert et al., 1969 b, 1973; Frances et al., 1970); and invertebrates (Boche and Quilligan, 1959; Wynder et al., 1963, 1965; Walker and Kiefer, 1966; Dalhamn, 1967; Weiss, 1968; Krahl and Bulmash, 1969; Kennedy and Elliott, 1970). Ciliary activity consistently shows a depression by cigarette smoke but bronchial clearance is influenced in a variable manner, the latter by ciliary activity and mucus formation, as discussed in Section III.

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VII. CARDIOPULMONARY FUNCTION IN SMOKERS AND NONSMOKERS

This section is a continuation of Section VI on the differences between smokers and nonsmokers. Measurements of cardiopulmonary function include ventilation-perfusion relationship, pulmonary circulation, cardiac function, pulmonary function, surfactant and other constituents of the lung. It is important to point out that animal experiments have served to clarify the mode of action by which the constituents of cigarette smoke influence the heart and lungs. The following bibliographic lists contain articles on general aspects of the topic under consideration.

No. 14. Constituents of cigarette smoke.

No. 15. Cardiopulmonary effects of cigarette smoke.

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VII-A. VENTILATION-PERFUSION RELATIONSHIPS

Measurement of pulmonary diffusion capacity shows a reduction in smokers compared with nonsmokers: Martt (1962) reporting from Missouri; Krumholz et al. (1964, 1965) from Indiana; Rankin et al. (1965) from Wisconsin; and Van Ganse et al. (1972) from the Netherlands. Anderson and Shephard (1969) reported from Canada that among 43 healthy adult men, by serial multiple regression analysis, pulmonary diffusion capacity correlated with age and weight. By using the predicted equation for diffusion coefficient, smoking habits had no substantial effect on diffusion capacity. Bates (1955) and McGrath and Thomson (1959) arrived at a similar conclusion with a limited number of subjects. It has also been suggested that the presence of carboxyhemoglobin in blood of smokers would account for the lower value for diffusion capacity.

Cigarette smokers show some inadequacy of gas mixing in the lung as compared with nonsmokers (Ross et al., 1967; Stănescu et al., 1968). There is also an alteration in the ventilation-perfusion ratio in smokers (Williams and Anderson, 1968 a, b; Anderson and Williams, 1969). The change has not been confirmed by others and the participation of pulmonary blood vessels in the phenomenon has not been proven.

Inhaling cigarette smoke by the use of a smoking device that brought more smoke into the lungs with each breath than would be the case in ordinary smoking did not significantly change blood gas exchange in normal subjects or patients with a moderate degree of emphysema (Motley and Kuzman, 1958). However, patients in California with severe emphysema showed decreases in arterial oxygen tension and saturation.

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VII. CARDIOPULMONARY FUNCTION IN SMOKERS AND NONSMOKERS

A. VENTILATION-PERFUSION RELATIONSHIPS

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VI. BRONCHOPULMONARY FUNCTION IN SMOKERS AND NONSMOKERS

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